PROBLEM FRACTURES – THE PHYSIOTHERAPY APPROACH

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INTRODUCTION

Most Chartered Physiotherapists deal with musculo-skeletal injuries.....or do they?

There is no doubt that MSk physios deal with structures that attach to the skeleton – muscles, ligaments, tendons, joint capsules etc., but very few actually ever treat the skeleton per se when it becomes injured.

We will be discussing what types of modalities can be used to treat the problem skeletal injuries – i.e. delayed or non-union fractures. We will discuss the different types of non-union, why they continue to be non-union and how physiotherapists can influence the healing rate of such non-unions by the use of certain treatment modalities which have extensive evidence to support their efficacy.

PHYSIOLOGY AND STRUCTURE

Before being able to treat bone, clinicians should re-acquaint themselves with the physiology of bone and the manner by which physiotherapists can influence that physiology to the benefit of the patient and the satisfaction of the orthopaedic surgeon.

Bone is the only body structure requiring longitudinal compressive strength as well as the ability to be able to withstand lateral and rotational forces. If bone were

constructed as a solid block it would be able to absorb longitudinal compression but would easily snap whenever lateral or rotational forces were applied – e.g. coral or a stick of Blackpool rock. Osteopetrosis and pycnodysostosis are both congenital conditions involving very dense, abnormal bones which fracture very readily and heal badly –e.g. Toulouse-Lautrec. However, bone consists mainly of hydroxyapatite – a bi-crystalline form of calcium phosphate and collagen fibres.

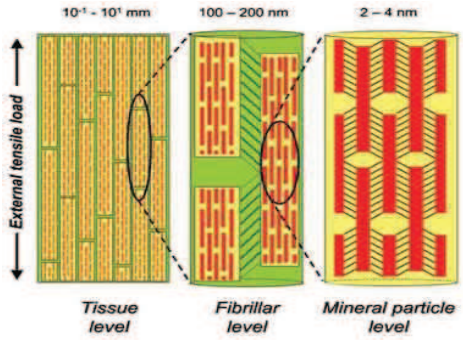
Bone responds to compression forces by generating a piezo-electric flow of current along the length of the bone. This current flow stimulates osteoblasts to deposit more bone along the track of the current, enabling bones to thicken in response to increased load e.g. weight gain, change in sporting activity levels etc.

Bone at the nanoscale

- When stress/pressure is applied to bone, this is absorbed by soft layers at successively lower length scales and less than 20% is noticed in the mineral phase. This strain induces a piezo-electric current which is vital for re-modelling and bone maintenance. NWB reduces this potential – osteopaenia.
- The soft structures (collagen) form a single rigid unit at the next level and so on, enabling the tissues to sustain large strains. Hence, brittle apatite remains shielded from excessive loads and does not break.
- Mineral crystallites are, nonetheless, very strong, capable of carrying more than 2-3 times the fracture load of bulk apatite.
- *(Max Planck Inst. And European Synchrotron Radiation Facility*
- *Compare with glass fibre mat, epoxy resin and GRP.*

Yellow cylinders = mineralised collagen fibrils in longitudinal section

Red tablets = mineral apatite crystallites embedded within the collagenous matrix of the fibrils



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Bone exists as :-

1) **Cortical bone** – which has high density, low surface area and low re-modelling rates. It is mostly present in the femoral neck and shafts of long bones.
2) **Trabecular bone** – which has low density, high surface area and high re-modelling rates and is mostly present in vertebral bodies and calcaneum.
3) **Periosteum** – is very important for normal vascularity and is an important source of bone-forming cells. Periosteum is very well innervated.

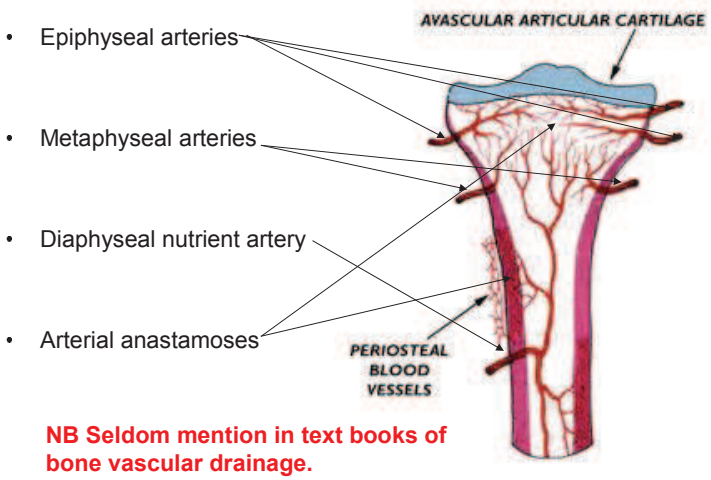
4) **Osteoblasts** – are cuboidal and mono-nucleated with basophilic cytoplasm. Their role is to form new bone. NB they exhibit a strong histochemical reaction for Alkaline Phosphatase (ALP). When mature, they synthesise Type 1 collagen but in non-unions they express Type III collagen. **2** They are attracted to, and migrate to, areas of bone with a –ve net potential.
5) **Osteoclasts** – are multi-nucleated and are responsible for bone resorption. They test strongly for Tartrate Resisitant Acid Phosphatase (TRAP) and are attracted to, and migrate to, areas of bone with net +ve electrical potential. Osteoclast apoptosis is delayed by oestrogen deficiency.
6) **Stromal cells** – produce growth factors for haemopoiesis and help the formation of osteoclasts as well as having the ability to differentiate to osteoblasts,

Bone allows the flow of blood and lymph through the medulla and Haversian vessels and also via the periosteum. Disruption of this blood supply and failure to re-establish a normal circulatory flow through the fracture is the principal reason for delayed and non-union.

Blood supply to bone.

- The skeleton receives **10-20%** of the cardiac output.
- Blood vessels especially rich in areas containing red bone marrow.
- Bone drains via veins that accompany the arteries and through foramina near the articular ends of bones i.e. haemopoietic region.
- Lymph vessels are abundant in the periosteum **BUT new research suggests lymph vessels are much more extensive and vital for drainage. (*Prof. Tony Pohl) 3***
- Because bone has no ability to accommodate swelling, impairment of drainage can result in the ultimate compartment syndrome.
- Vascular stasis results in local hypoxia or even anoxia with tissue necrosis (similar to varicose ulcers)
- Reduced drainage causes metabolite accumulation – H_2CO_3, pyruvic acid, lactic acid etc. which may attract acidophilic cells i.e. osteoclasts and produce a hostile environment for basophilic osteoblasts.

Blood supply to bone



Reasons for Non-Union

It is well known that fractures in poorly vascularised bones such as scaphoid, neck of talus and the junction of the middle and lower thirds of the tibia have a much greater tendency to progress to non-union. High speed trauma may involve extensive displacement at the fracture site with resultant periosteal stripping and further vascular damage. Pollution of bone ends in compound fractures may introduce pathogens which can further interfere with blood supply.

Types of Non-Union

Hypertrophic- extensive callus formation with good vascularity but no complete bridging. This type of non-union probably is due to inadequate immobilisation. These respond well to proper stabilisation and good therapeutic management.

Atrophic- absence of callus and atrophic bone ends which may appear tapered and osteopaenic, but are sometimes sclerotic. Bone vascularity is very poor with poor healing potential. There are two sub-types-

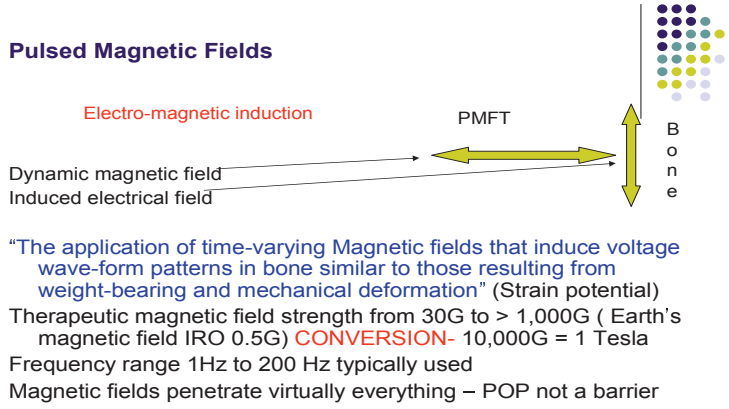
- **Simple atrophic**- has the potential to respond to treatment to encourage angio-neo-genesis

- **Synovial atrophic-** forms a fibrous fluid filled capsule around the bone ends and is referred to as a synovial pseudarthrosis. Bones such as the clavicle can comfortably tolerate a synovial pseudarthrosis with little pain or reduction in function. Non-surgical treatment of synovial pseudarthrosis will almost certainly not achieve bony union. Removal of the capsule and freshening of the bone ends is essential.

Normatrophic- both ends have moderate vascularity and have moderate healing potential. This type of non-union is eminently suitable for treatment by several modalities.

TREATMENT MODALITIES

Pulsed Magnetic Field Therapy (PMFT) in the treatment of fractures is defined as the application of time varying magnetic fields that induce voltage wave form patterns in bone similar to these resulting from mechanical deformation. **4** The voltage is induced at right angles to the pulsed or dynamic magnetic field. Magnetic field strength in clinical application ranges from 30G to 1000G (the earth's magnetic field is up to 0.5G) and with a frequency range of 1-200Hz. PMFT was approved by the FDA in 1979 for the treatment of fractures and non-union fractures.



A literature review by Gossling et al in 1992 **13** concluded that PMFT alone was at least as effective as surgery in cases of non-union with success rate of 80% +.

Mooney in 1990, **5** studying the efficacy of PMFT in lumbar inter-body fusions showed a success rate of 92% in the PMFT group compared to 65% in the non-treated, placebo group. A similar study by Marks in 2000 **6** showed 97.6% fusion rate with PMFT compared to 52.6% in the placebo group.

Bassett et al in 1982 **7** showed in a group of patients with an average of 4.7 years of non-union, 3.4 previous surgical failures and 35% infection rate, bone healing took place in 75% of patients.

The effects PMFT observed in bone in fracture non-unions and failed arthrodeses involved angiogenesis, increased mineralisation, increase in endochondral ossification, increase in osteoblastic activity and decrease in osteoclastic activity.

PMFT penetrates anything, including POP and Baycast. Metal implants do not present a problem since PMFT's are non-thermic.

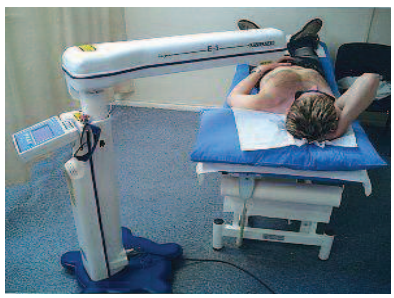
LASER

Therapeutic lasers are coherent, mono-chromatic light emissions. Some laser diodes have up to 6 degree divergent beams, thus obeying the Inverse Square Law but many more powerful scanning lasers have non-divergent beams.

Infra-red lasers

Usual frequencies 630nM (visible) – 980nM.

Intensities 5mW hand-held pen laser (Class IIIB) to 4W scanning laser (Class IV)



Therapeutic wavelengths range from 600nm to 1000nm. Outputs range from 5mW to 3000mW with treatment times varying from mere seconds to several minutes.

Dickson et al **8** showed a 300% increase in ALP expression (indicator of osteoblast activity) in rat femoral fractures. The main effect is photo chemical and not thermic.

Yaakobi and Oron **9** showed that 630nm irradiation of the rat tibia caused a 120% increase in ALP and a 40% reduction in TRAP, and that at 13 days reparative compact bone was measured at 88% against 44% in non-irradiated animals.

Low Intensity Pulsed Ultra-Sound – LIPUS

LIPUS uses an output of about 1MHz at an intensity of 20mW/cm2 up to 200mW/cm2. LIPUS was approved by the FDA in 1994 for the accelerated healing of fresh fractures and in 2000 for the healing of established non-union fractures. The studies presented to the FDA showed that LIPUS had a positive effect by enhancing angiogenesis, chondrogenesis and osteogenesis.

Low Intensity Pulsed Ultra Sound - LIPUS

Gross et al in 1997 **10** suggested that, as LIPUS energy is absorbed at a rate proportional to tissue density, gradients of mechanical stress would be produced when applied to a callus where cartilage/bone/collagen interfaces abound. Wallace

et al in 1994 **14** showed that there was stimulation of vascular activity as part of a bone's response to mechanical stress.

World-wide clinical studies using LIPUS for the treatment of non-unions demonstrated a healing rate of 88% in non-unions presenting with an average of 23 months since fracture. When compared with surgery, LIPUS for non-unions has the same success rate as surgery.

The study by Heckmann et al **11** in 1994 on delayed union tibial fractures showed only 6% progressing to full non-union compared to 36% non-unions in the control group.

HYPERBARIC OXYGEN THERAPY (HBOT)

HBOT involves breathing100% medical oxygen while lying or sitting in a compression chamber in which the surrounding pressure is at about double normal atmospheric pressure. Normal air is about 20% O_2, so breathing 100% O_2 at double atmospheric pressure can cause up to a 10 fold increase in O_2 levels dissolved in the plasma – NOT in red blood cells. Henry's Law states that "the concentration of a gas in a fluid is determined by pressure and the solubility of the gas". RBCs supply O_2 directly to brain cells and muscle fibres but most cells are oxygenated by dissolved O_2 in the plasma. Lack of O_2 is thought to be a limiting factor in fracture healing. Multipotential precursors of fibroplastic origin form cartilage which is relatively avascular in the presence of low pO_2 but form bone when tissue pO_2 is elevated. **12**

Hyperbaric oxygen therapy

Effects of HBO

2ATA (10 metre depth)+ 100% O_2.

Peripheral vaso-constriction with capillary "twigging".

Raising of plasma O_2 levels by up to 10X.

Angioneogenesis with raised O_2 in plasma reduce extent of necrosis.

Mitochondria utilise 80% of the molecular O_2 with the other 20% being taken up by microsomes, nuclei and plasma membranes. In trauma, where local blood vessels have been destroyed, cells in the centre/ epicentre of the fracture can become hypoxic or anoxic. Under normal circumstances, the rate of utilisation of O_2 is blood flow limited but, if damaged cells are beyond a critical distance from the nearest capillaries, then the O_2 utilisation becomes diffusion limited. ATP synthesis can no longer occur and mitochondrial function ceases. Healing cannot occur until O_2 supply is restored, either by angiogenesis or by otherwise increasing the O_2 in the plasma which continues to perfuse the fracture.

It is accepted that most physios would not have access to HBOT.

SUMMARY

A wound is defined as an injury to the body which causes a disruption in the normal continuity of the body structures. Fractures may be defined as the disruption of continuity of bone and yet, while physios would expect to successfully treat wounds in other tissues, they have traditionally drawn a line at trying to create an ideal healing environment for bone injuries.

If the aim of treatment is to reduce tissue oedema and thus lower interstitial pressure, the result is improved vascular flow, increased cellular pO_2 and improved rate of healing. If the aim of treatment is angiogenesis then there are many options available in the physiotherapist's armamentarium to achieve that aim. If the aim of treatment is to create an optimal physiological environment for healing, then physiotherapists have been doing just that for years when treating muscles, ligaments, tendons, skin etc and, with the cooperation of surgeons have the capability to do precisely the same with bone wounds – i.e. fractures.

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